Type: Oral Presentation

Final Abstract Number: 58.039
Session: Bacterial Infections
Date: Saturday, April 5, 2014
Time: 12:45-14:15
Room: Ballroom

Diabetic patients are threatened by "superbugs" *Acinetobacter baumannii* In Saudi Arabia hospitals

A.A. Al-Sultan

King Faisal University, Alahsa, Saudi Arabia

Background: One of the greatest threat to the recent generation of antibiotics is the increasing prevalence of bacterial resistant, specially Gram-negative bacteria. *Acinetobacter baumanni* is one of the most important superbugs becoming increasingly prevalent in patients with diabetes mellitus in Middle East particularly in Saudi Arabia. We have shown that diabetic patients are more susceptible to acquire infection with these type of superbugs *A.baumannii* than the rest of the population. Carbapenem resistance in *A.baumannii* limits therapeutic options and is largely manifested by β–lactamases and metallo-β-lactamase that play a significant role in mechanisms of drug-resistance in diabetic patients. The aim is to investigate the level of threat of these pathogens to diabetics and the mechanisms of drug-resistance of patients in Saudi Arabia hospitals.

Methods & Materials: A total of 64 non-repetitive, strains collected between 2008 to 2012 from different specimens from diabetic patients from King Faisal Specialist Hospital and Research Centre, Riyadh. All isolates were identified by Vitek system. Multiplex PCR using primers for $bla_{\text{OXA-51}}$ combined with primers for $bla_{\text{OXA-23}}$, $bla_{\text{OXA-24/40}}$ and $bla_{\text{OXA-58}}$ was employed. PCR was used for detection of β-lactamase and metallo-β-lactamase genes and insertion sequences IS elements. Pulsed-field gel electrophoresis (PFGE) has been performed. MICs was determined by dilution method.

Results: Sixty-four clinical isolates of *A. baumannii* having $bla_{\text{OXA-51-like}}$ gene. All isolates were resistant to imipenem and meropenem (MIC > 16 - 32 mg/L) except four were intermediate (MIC = 8 mg/L) and Six were susceptible (MIC 0.5 - 2 mg/L) to both of them. All isolates were sensitive to tigecycline and colistin (MIC 0.5 - 2 mg/L) except four were resistant (MIC 8-32 mg/L) and three were intermediate to tigecycline only (MIC 4 - 8). The cause of resistance was from β-lactamase of $bla_{\text{OXA-23}}$ and $bla_{\text{OXA-24/40}}$ and metallo-β-lactamase bla_{VIM} and bla_{SPM}. Fifty-nine isolates were possess the insertion sequence IS*Aba*1. PFGE showed Nine clusters of $bla_{\text{OXA-51-like}}$ enzymes.

Conclusion: Carbapenem-resistant *Acinetobacter baumannii* is a serious superbugs threaten diabetic patients due to the circulation of β-lactamases and metallo-β-lactamase in Saudi Arabian hospitals. Furthermore, the emergence of tigecycline plus colistin resistance is causing a treatment shortfall for these patients.

http://dx.doi.org/10.1016/j.ijid.2014.03.1144

Type: Poster Presentation

Final Abstract Number: 58.040
Session: Bacterial Infections
Date: Saturday, April 5, 2014
Time: 12:45-14:15
Room: Ballroom

Genome sequence analysis of a Belgian epidemic cystic fibrosis *Pseudomonas aeruginosa* isolate

J. Dingemans [1], F. Hildebrand [1], L. Ye [1], M. Craggs [1], F. Tontodonati [1], D. De Vos [2], J.-P. Pirnay [2], A. Crabbé [3], R. Van Houdt [4], A. Malfroot [5], P. Cornelis [1]

[1] *Vrije Universiteit Brussel, Brussels, Belgium*
[2] *Queen Astrid Military Hospital, Brussels, Belgium*
[3] *Arizona State University, Tempe, Arizona, USA*
[4] *Belgian Nuclear Research Centre, SCK•CEN, Mol, Belgium*
[5] *Universitair Ziekenhuis Brussel, Brussels, Belgium*

Background: *Pseudomonas aeruginosa* chronically infects cystic fibrosis (CF) patients leading to persistent lung inflammation and, ultimately, death of the patient. In a previous study we have detected an epidemic *P. aeruginosa* strain amongst patients attending the CF reference center in Brussels and three other Belgian CF reference centers. This strain (next to other *P. aeruginosa* CF strains) was characterized by a deletion of the *fpvB* gene, involved in ferripyoverdine uptake.

Methods & Materials: In order to identify deletions, acquired during colonization of *P. aeruginosa* in the CF lung, we decided to sequence the complete genome of the epidemic *isolate P. aeruginosa* CF_PA39. In this study, we analyzed the genome sequence of this isolate using a combination of bioinformatic tools such as RAST, IslandViewer, PHAST, and CRISPRFinder.

Results: Interestingly, large deletions were detected comprising the entire type III secretion region (ca. 24 kbp) and the region corresponding to PA2171-PA2226 of the PAOI reference genome (ca. 58 kbp) harboring several amino acid transport, virulence, and resistance genes. Furthermore, we have found ten different genomic islands, including well-described (LESGI-2, LESGI-4, PADK2 GI-2, PA7 RGP 69, and PA7 RGP 76) and novel sequences. Interestingly, using the genome sequence of this epidemic strain, we have identified an open reading frame (ORF) encoding a potentially novel S-type pyocin. This novel pyocin sequence perfectly matched to the nucleotide sequences of both the receptor binding and translocation domains of pyocin S1. However, the nucleotide sequence corresponding to the killing domain of this novel pyocin sequence was completely different compared to that of pyocin S1. Using RT-PCR, we showed that the expression of the pyocin gene is induced under iron-limiting conditions. Finally, we have proven the functionality of this pyocin as it was able to inhibit the growth of a number of *P. aeruginosa* CF isolates.

Conclusion: In summary, by analyzing the genome sequence of a highly adapted CF isolate, we gained insight in the adaptation of *P. aeruginosa* to the CF lung at the genomic level and discovered a novel, hybrid, S-type pyocin, termed pyocin S6. Moreover, this study elaborates on the still contentious issue of highly transmissible clones among CF patients.

http://dx.doi.org/10.1016/j.ijid.2014.03.1145